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Definition of the interacting interfaces of Apobec3G and HIV-1 Vif using MAPPIT mutagenesis analysis

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Background

The host restriction factor Apobec3G is a cytidine deaminase that incorporates into HIV-1 virions and interferes with viral replication. The HIV-1 accessory protein Vif subverts Apobec3G by targeting it for proteasomal degradation. We studied the Apobec3G homomerisation and the interaction of Apobec3G with Vif in detail.

Methods

We used the MAPPIT two-hybrid technique to analyse the Apobec3G-Apobec3G and the Apobec3G-Vif interactions in intact human cells. MAPPIT is based on the functional complementation of a cytokine receptor signalling pathway.

Results

We propose a model in which Apobec3G N-terminal domains symmetrically interact via a head-to-head interface containing residues 122 RLYYFW 127. Mutations in the head-to-head interface abrogate the Apobec3G-Apobec3G interaction. All mutations that inhibit Apobec3G-Apobec3G binding also inhibit the Apobec3G-Vif interaction, indicating that the head-to head interface plays an important role in the interaction with Vif. Only the D128K, P129A and T32Q mutations specifically affect the Apobec3G-Vif association. In our model, D128, P129 and T32 cluster at the edge of the head-to-head interface, possibly forming a Vif binding site composed of two Apobec3G molecules.

Discussion

We propose that Vif either binds directly at the Apobec3G head-to-head interface or associates with an RNA-stabilized Apobec3G oligomer.

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